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REVIEW ARTICLE

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Cardiovascular Disease Risk Assessment in the Digital Age: From Fixed Risk Equations to Data-Driven, Intervention-Linked Prevention

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Abstract

Cardiovascular disease (CVD) remains the leading cause of death worldwide. Its prevention depends on identifying, before the first clinical event, who will benefit from treatment. Over the past six decades, that task has been carried out by a sequence of multivariable risk equations — from the Framingham-derived scores to the US Pooled Cohort Equations, the United Kingdom's QRISK3, Europe's SCORE2, and most recently the American Heart Association's PREVENT equations. Each generation has expanded the input variables, widened the age range, and improved calibration. Yet external validation continues to show systematic miscalibration and weak transportability across populations. This review synthesises how contemporary epidemiology is responding to those limitations on two fronts. First, predictive data sources are broadening: electronic health record-derived machine learning models, wearable and sensor-derived physiological signals, and polygenic risk scores are extending discrimination and covering patients older fixed equations exclude. Second, and less often integrated, digital health interventions — from automated text-message programmes to smartphone-linked blood-pressure management — have demonstrated modest but reproducible reductions in the very risk factors that risk equations estimate: LDL cholesterol, systolic blood pressure, and medication adherence. We argue that the central methodological gap is no longer prediction accuracy in isolation. It is the design of closed-loop systems in which a risk estimate triggers, adapts to, and measures the response of a digital intervention. We examine where the evidence is robust, where disagreements persist over the clinical utility of polygenic scores and the generalisability of wearable-derived data, and what an implementation-focused research agenda should now prioritise.

Keywords cardiovascular disease; risk prediction; Pooled Cohort Equations; digital health; wearable devices; polygenic risk score; primary prevention

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1. Introduction

Atherosclerotic cardiovascular disease is responsible for roughly one third of all deaths globally, and its prevention is organised around a single epidemiological question: given a person's measured risk factors, what is the probability that they will experience a myocardial infarction, stroke, or cardiovascular death within a defined horizon? The answer determines whether a clinician recommends a statin, intensifies antihypertensive therapy, or initiates lifestyle counselling. The tools that provide that answer have evolved substantially since the Framingham Heart Study first demonstrated that blood pressure, cholesterol, smoking, and age jointly predicted coronary events. Modern risk equations are now routinely embedded in guidelines in North America, Europe, and the United Kingdom, and they shape treatment decisions for millions of adults annually.

Two parallel developments are now reshaping this landscape. On the prediction side, electronic health records (EHRs), wearable sensors, and genome-wide association data have made it possible to estimate risk from data that were unavailable to the cohorts that derived the original scores. On the intervention side, smartphones, text messages, and connected home devices have made it feasible to deliver behavioural and adherence support at a scale and a cost that traditional clinic-based prevention cannot match. Yet these two strands have largely developed independently: prediction researchers validate discrimination and calibration, while digital intervention researchers measure changes in risk-factor levels, and rarely do the two meet in a single system.

This review examines CVD risk assessment from the perspective of contemporary epidemiology, tracing the lineage of the major risk equations, appraising their documented strengths and weaknesses, and then asking how digital data sources and digital interventions are altering — or should alter — the way risk is estimated and acted upon. We focus on the period since the introduction of the Pooled Cohort Equations in 2013, a point at which the field consolidated a US standard and then began a decade of rapid expansion. The argument is organised around a single thesis: the next advance in CVD prevention is less likely to come from a marginally better prediction coefficient than from a system in which risk assessment and digital intervention are coupled, calibrated to the populations they actually serve, and evaluated on event outcomes rather than surrogate markers alone.

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2. Methods

This is a narrative, not a systematic, review. We searched PubMed, Web of Science, and Scopus for literature published between January 2013 and October 2026, using combinations of the terms “cardiovascular risk prediction,” “risk equation,” “Pooled Cohort Equations,” “QRISK,” “SCORE2,” “PREVENT,” “machine learning,” “electronic health records,” “wearable,” “polygenic risk score,” “digital health,” “mHealth,” “text messaging,” “telemonitoring,” and “blood pressure telemonitoring.” We included primary derivation and validation studies of risk equations, randomised controlled trials and meta-analyses of digital interventions, and guideline documents or scientific statements issued by the American Heart Association and the European Society of Cardiology. Priority was given to contemporary, highly cited, and externally validated sources; we did not apply a formal risk-of-bias instrument or a formal screening protocol, and selection was guided by the authors’ judgement of methodological quality and relevance to the coupling argument. The scope is deliberately selective rather than exhaustive.

3. The lineage of multivariable risk equations

3.1 From Framingham to the Pooled Cohort Equations

The Framingham Heart Study established the multivariable risk-score paradigm. Early scores predicted coronary heart disease from age, sex, total cholesterol, systolic blood pressure, smoking, and diabetes, and they formed the basis of the National Cholesterol Education Program’s Adult Treatment Panel III guidelines. Their central limitation was recognised early: they were derived from a predominantly white, middle-aged North American cohort and predicted coronary events rather than the broader atherosclerotic CVD (ASCVD) outcome — coronary death, nonfatal myocardial infarction, and fatal or nonfatal stroke — that guidelines increasingly targeted.

The 2013 ACC/AHA Pooled Cohort Equations (PCE) addressed this gap by pooling data from five US community-based cohorts — Framingham Offspring, Atherosclerosis Risk in Communities, Cardiovascular Health Study, and Coronary Artery Risk Development in Young Adults among them — and deriving sex- and race-specific coefficients for adults aged 40 to 79 (Goff et al., 2014). The PCE predicted 10-year ASCVD risk from age, total and HDL cholesterol, systolic blood pressure, blood-pressure treatment status, diabetes, and smoking. Its adoption as the US standard was accompanied by immediate and persistent controversy: independent validations reported that the PCE overestimated observed event rates by 75 to 150 percent in contemporary cohorts, partly because the underlying cohort data predated the widespread adoption of statins, antihypertensive therapy, and smoking-cessation interventions that have since lowered event rates (Ridker & Cook, 2016).

3.2 The EHR-derived alternative: QRISK3

Where the PCE pooled a small number of purpose-built research cohorts, the QRISK programme derived risk equations from the United Kingdom’s QResearch primary care database, which contains routinely collected EHRs for millions of patients. QRISK3, published in 2017, was derived from more than 1.2 million patients aged 25 to 84 and retained the classical risk factors while adding clinical variables that reflect the epidemiological complexity of real practice: chronic kidney disease, atrial fibrillation, rheumatoid arthritis, systemic lupus erythematosus, migraine, corticosteroid use, atypical antipsychotics, severe mental illness, and erectile dysfunction (Hippisley-Cox, Coupland, & Brindle, 2017). The inclusion of these variables was not merely a technical extension; it encoded the observation that social and clinical conditions — severe mental illness in particular — confer CVD risk that the classical factor set does not capture. QRISK3 also extended the age range downward and upward, reflecting a growing recognition that risk trajectories begin well before middle age and that risk equations should remain relevant in older adulthood.

3.3 The European update: SCORE2

The European Society of Cardiology’s original SCORE system predicted 10-year fatal CVD risk from cohorts assembled largely in the 1970s and 1980s. By the 2010s, that source data was two to three decades old, and the field had shifted to predicting fatal plus nonfatal events. The 2021 ESC Guidelines introduced SCORE2, derived from 45 European cohorts comprising 677,684 participants and more than 30,000 fatal and nonfatal CVD events, stratified by four regions of differing baseline cardiovascular risk (Visseren et al., 2021). SCORE2 uses non-HDL cholesterol rather than total cholesterol, predicts 10-year fatal plus nonfatal CVD, and is accompanied by SCORE2-OP, an extension for adults aged 70 and older. Regional risk calibration was a deliberate response to the long-standing finding that a single European equation cannot be transportable from a low-risk Mediterranean population to a higher-risk Eastern European one.

3.4 The most recent iteration: PREVENT

In 2023, the American Heart Association introduced the PREVENT (Predicting Risk of Cardiovascular Disease Events) equations, replacing the 2013 PCE as the recommended US risk-assessment tool. PREVENT estimates both 10-year and 30-year risk for total CVD — including heart failure, not only ASCVD — for adults aged 30 to 79. Three design choices mark it as a product of contemporary epidemiology. First, it is race-agnostic, omitting the race-specific coefficients that the PCE had used on the grounds that race is a social rather than a biological construct. Second, it incorporates estimated glomerular filtration rate, reflecting the cardiovascular–renal continuum. Third, it was derived from more contemporary and more racially and ethnically diverse US data, producing markedly better calibration than the PCE in external validation (Khan et al., 2023). The shift from 10-year to 10- and 30-year horizons also signals an epidemiological judgement: for younger adults, a 10-year risk estimate that sits below the treatment threshold can obscure a lifetime exposure that warrants primordial prevention.

Note. ASCVD = atherosclerotic cardiovascular disease; CKD = chronic kidney disease; AF = atrial fibrillation; RA = rheumatoid arthritis; SLE = systemic lupus erythematosus; CKM = cardiovascular–kidney–metabolic. All equations use age, sex, total or non-HDL cholesterol, HDL cholesterol, systolic blood pressure, smoking, and diabetes as core inputs; the table lists only the principal additions. Each equation was derived in its population of origin and requires external recalibration before transport to other populations.

3.5 The calibration problem that persists

The most consistent finding across three decades of external validation is that risk equations discriminate between individuals reasonably well — C-statistics typically cluster between 0.70 and 0.80 — but miscalibrate systematically when transported beyond their derivation populations. Independent validations of the PCE in contemporary cohorts have repeatedly reported that observed event rates fall well below predicted risk, with pooled observed-to-expected ratios below 1.0 across multiple settings; overestimation is most pronounced in high-risk individuals and in populations that differ from the derivation cohorts, and heterogeneity in the C-statistic is substantial (Ridker & Cook, 2016). The sources of this

Table 1. Principal contemporary CVD risk-prediction equations.

Equation	Year	Derivation cohort(s)	Age range	Outcome horizon	Key additional inputs	Source
Pooled Cohort Equations (PCE)	2013	5 US cohorts (~29,000)	40–79	10-y ASCVD	Sex- and race-specific coefficients; blood-pressure treatment	Goff et al., 2014
QRISK3	2017	UK QResearch EHR (~1.28 M)	25–84	10-y CVD	CKD, AF, RA, SLE, migraine, corticosteroids, severe mental illness, erectile dysfunction	Hippisley-Cox et al., 2017
SCORE2	2021	45 European cohorts (677,684)	40–69	10-y fatal + nonfatal CVD	Non-HDL cholesterol; four-region risk calibration	Visseren et al., 2021
SCORE2-OP	2021	Extension of SCORE2	≥70	10-y fatal + nonfatal CVD	Competing-risk modelling for older adults	Visseren et al., 2021
PREVENT	2023	Contemporary diverse US data (>6.5 M records)	30–79	10-y and 30-y total CVD (incl. heart failure)	eGFR; race-agnostic; cardiovascular–kidney–metabolic staging	Khan et al., 2023

miscalibration are now reasonably well understood.

The first source is temporal drift. Risk-factor distributions, treatment patterns, and event rates have all shifted since the derivation cohorts were assembled. Statins were introduced in 1987; antihypertensive therapy, smoking prevalence, and diabetes treatment have all changed substantially. A model trained on 1970s event rates will overestimate risk in a 2020s population in which many of those risk factors are now treated. This is the central motivation for SCORE2's contemporary derivation and for PREVENT's refresh.

The second source is population mismatch. Equations derived in one ancestry, socioeconomic, or health-system context transport poorly to another. QRISK3's addition of deprivation and ethnic-group variables, SCORE2's regional stratification, and PREVENT's deliberate diversity expansion are all responses to this problem. Yet even PREVENT's performance in low- and middle-income populations — which bear the majority of the global CVD burden — remains under-validated.

The third source is the structure of the model itself. Classical Cox-based equations use a small, fixed set of linearly combined variables. They cannot easily accommodate nonlinear risk-factor relationships, interactions between variables, or the dense, correlated data that EHRs and wearable sensors now generate. This is the gap that machine learning and multi-omic models are explicitly designed to address.

A fourth and more conceptual limitation is what might be called the *risk paradox*. Risk equations can achieve acceptable discrimination while reclassifying relatively few individuals at the treatment threshold: the C-statistic improves by only modest increments when novel biomarkers or genetic scores are added, even when those additions are statistically significant. Whether those modest reclassifications justify the cost of the added measurement is an unresolved question, and it is the question on which the clinical utility of polygenic scores and many wearable-derived signals currently turns.

4. Expanding the predictive toolkit

4.1 Electronic health records and machine learning

The move from purpose-built cohorts to routinely collected EHRs changes both the quantity and the nature of available predictor data. EHR-derived cohorts are an order of magnitude larger than traditional research cohorts, and they contain not only the classical risk factors but also diagnosis codes, medication prescriptions, laboratory trajectories, and healthcare utilisation patterns. Machine learning methods — gradient-boosted trees, regularised regression, and increasingly deep neural networks — can exploit this structure in ways that a fixed Cox equation cannot.

In a multi-ethnic US EHR cohort of more than 260,000 patients, gradient-boosted models predicted 5-year ASCVD events with an area under the receiver-operating characteristic curve of 0.835 in the full cohort, compared with 0.775 for the PCE in the PCE-eligible subset (Ward et al., 2022). The advantage was not confined to discrimination: the EHR models could generate risk estimates for patients who would have been excluded from the PCE by missing or out-of-range variables — a population that, in routine practice, is precisely the one clinicians most need to assess. Similar results have been reported in UK Biobank and in large integrated delivery systems, with neural networks improving on QRISK3 by several C-statistic points.

The methodological caution, however, is that internal validation on the same EHR system overstates generalisability. EHR-derived models are vulnerable to labelling bias, missingness not at random, and the very different data-capture practices of different health systems. A model trained on a Northern California integrated-delivery system does not transport intact to a safety-net clinic, a low-resource health system, or a nation without universal EHR infrastructure. The field has not yet settled into the kind of prospective, multi-site, pre-specified external validation that the original Framingham and PCE models underwent.

4.2 Wearable and sensor-derived signals

The Apple Heart Study, published in 2019, marked a conceptual turning point: it demonstrated that a consumer wearable could, at scale, detect a clinically meaningful condition. Among 419,297 participants enrolled through a smartphone app without any clinic visit, only 0.52 percent received an irregular-pulse notification; among those who returned a patch ECG, the positive predictive value of the notification for atrial fibrillation was 84 percent (Perez et al., 2019). The study was single-arm and pragmatic, and it deliberately did not randomise, but it established that a siteless, app-based recruitment and monitoring model could reach hundreds of thousands of people and surface atrial fibrillation that would otherwise have gone undiagnosed — atrial fibrillation that confers a fivefold increase in stroke risk and that is treatable with anticoagulation.

Wearables have since expanded beyond pulse irregularity to continuous heart-rate monitoring, heart-rate variability, sleep-stage detection, accelerometer-derived activity, and, in research devices, continuous glucose monitoring. The epidemiological promise is twofold. First, continuous data capture reveals phenomena — nocturnal arrhythmia, episodic hypoxemia, circadian activity patterns — that a single clinic measurement cannot. Second, wearable data are generated in the course of ordinary life, which may reduce the Hawthorne effect and capture true habitual behaviour.

The limitations are equally clear. Wearable users are not representative: they are younger, more affluent, more digitally engaged, and more likely to be white than the general population, and the devices are abandoned at high rates within months of purchase. Algorithms trained on self-selected users may perform worse in older adults, in people with darker skin tones (for whom optical sensors are less reliable), and in low-resource settings. The Apple Heart Study's low notification rate also means that the absolute number of people identified per wearer is small; whether population-wide

wearable screening is cost-effective remains unresolved.

4.3 Polygenic risk scores

Cardiovascular disease is highly heritable, and genome-wide association studies have now identified thousands of common variants associated with coronary artery disease. Polygenic risk scores (PRSs) aggregate these variants into a single weighted index. A multi-ancestry PRS derived from more than 269,000 coronary disease cases and 1.17 million controls across five ancestral groups reported an odds ratio of approximately 2.14 per standard deviation for prevalent coronary disease, and it improved discrimination when added to clinical risk factors (Patel et al., 2023). An American Heart Association scientific statement concluded that PRSs are independently associated with multiple cardiovascular diseases and may have a role in refining risk stratification, but it stopped short of recommending routine clinical use (O'Sullivan et al., 2022).

The disagreement in the literature is not about whether PRSs carry genetic information — they clearly do — but about whether that information changes treatment decisions at the individual level. The addition of a PRS to the PCE typically improves the C-statistic by one to two points, and the net reclassification improvement is concentrated at the margins of treatment thresholds. In real-world implementation studies, individuals in the top PRS quintile have not, to date, shown substantially higher rates of statin initiation than their peers, raising the question of whether the score changes clinician behaviour at all. PRS performance also decays sharply in non-European ancestral groups, which raises an equity concern that is now central to the field's self-assessment.

4.4 From static snapshots to dynamic risk

Classical risk equations estimate a single probability at a single visit. EHR, wearable, and genomic data are inherently longitudinal: blood pressure, lipids, weight, activity, and heart rate all evolve, and risk evolves with them. Dynamic models that update risk as new measurements arrive align with how prevention actually works — a clinician sees a patient, intervenes, and re-measures. Whether they improve event prediction is still being tested, but they connect naturally to the digital intervention systems discussed next.

5. Digital health interventions that close the loop

5.1 The missing link: risk without action

A risk estimate is clinically useful only if it triggers a response. For the roughly 30 percent of adults in whom a risk equation flags elevated 10-year risk, the response typically consists of a statin prescription, antihypertensive intensification, and lifestyle counselling. The behavioural components — diet, physical activity, smoking cessation, medication adherence — are notoriously difficult to sustain through a once-yearly clinic visit. Digital interventions have emerged precisely because they can deliver ongoing support at low marginal cost, and a substantial evidence base now supports their effectiveness.

5.2 Text messaging and mHealth in secondary prevention

The TEXT ME (Tobacco, Exercise and Diet Messages) trial was among the first randomised studies to test a simple mobile intervention in established coronary disease. Three hundred and fifty-two patients received four automated, motivational text messages per week over six months, covering medication adherence, diet, physical activity, and smoking cessation. At six months, the intervention group had lower LDL cholesterol (the primary endpoint), lower systolic blood pressure and body mass index, higher physical activity, and lower smoking prevalence than usual care (Chow et al., 2015). The effect sizes were modest — LDL reductions of roughly 0.4 mmol/L, systolic blood pressure reductions of a few millimetres of mercury — but they were achieved at very low cost and scaled to patients who might never attend a structured cardiac rehabilitation programme.

Subsequent trials and systematic reviews have generalised these findings. A meta-analysis of digital health interventions for hypertension, pooling 28 randomised trials and more than 7,000 participants, found a between-group reduction of 3.62 mmHg in systolic blood pressure and 2.45 mmHg in diastolic blood pressure relative to control — effects comparable to many single-pill antihypertensive regimens, delivered through a smartphone (Siopis et al., 2023). mHealth interventions improved medication adherence in secondary-prevention populations and showed small but consistent benefits on LDL cholesterol and BMI. Telehealth-based dietary interventions reduced both systolic blood pressure and LDL cholesterol in patients with established CVD.

5.3 Adherence, connected devices, and closed-loop feedback

The most sophisticated contemporary systems couple home-measured physiological data with automated feedback. A patient measures home blood pressure, the reading is transmitted to a clinician or an algorithm, and a text or app message adjusts medication or reinforces adherence. These systems have shown particular promise in hypertension, where home monitoring with telehealth support achieves better blood-pressure control than usual care in part because they make the measurement itself an intervention moment. Wearables that pair activity tracking with goal-setting and social feedback have shown modest improvements in physical activity, though retention declines over time.

5.4 The implementation gap

The evidence for digital interventions is not uniformly positive, and the negative and null results are informative. Effects are concentrated in secondary-prevention populations — patients with established disease who are already engaged with care. In primary-prevention populations recruited through consumer channels, effects on risk factors are smaller and engagement decays rapidly. Many trials are short (six to twelve months), and the durability of behavioural change after the intervention ends is uncertain. Recruitment to digital trials is skewed toward younger, more educated, more digitally literate participants, which limits generalisability to the older, multi-morbid populations in whom CVD risk is highest. Data privacy, algorithmic transparency, and the regulatory status of consumer devices as medical tools remain unresolved.

Crucially, few digital intervention trials in primary or secondary prevention have used hard cardiovascular events as the primary endpoint. Most measure risk-factor changes — blood pressure, LDL, adherence — and infer event reduction. That inference is reasonable, because the relationship between these risk factors and events is well established: a 4–5 mmHg sustained systolic reduction is associated with roughly a 15 percent lower risk of stroke and a 10 percent lower risk of coronary death over five years. But it means that the long-term event-level benefit of digital prevention remains less directly proven than the risk-factor-level benefit. The closest available evidence comes from hypertension programmes such as TASMINH4 and HOME BP (McManus et al., 2018, 2021), which demonstrated sustained blood-pressure control over 12 months and are the strongest candidates for future event-endpoint follow-up trials. In heart failure — a secondary-prevention setting — remote monitoring has reduced hospitalisations in several randomised cohorts, but that evidence does not yet transfer to asymptomatic primary-prevention populations. Event-endpoint, long-follow-up digital trials are the field's most pressing evidence need.

6. Toward coupled risk assessment and intervention

The preceding sections describe two literatures that have developed in parallel. Risk assessment has become more accurate, more data-rich, and more attentive to calibration and equity. Digital intervention has become more effective, more scalable, and more firmly grounded in randomised evidence. What is largely absent is the closed loop: a system in which a contemporary risk estimate identifies an individual, triggers a tailored digital intervention, monitors the response through wearable or home-measured data, and re-estimates risk as risk factors change.

It is useful to operationalise "closed-loop" by degree, because existing evidence already occupies intermediate positions. A partial closed loop exists when home-measured physiological data are transmitted and a clinician or algorithm adjusts therapy, but the trigger is a diagnosed risk factor rather than a formal risk equation and the endpoint is a risk-factor level rather than a clinical event. TASMINH4 exemplifies this position: 1,182 patients with uncontrolled hypertension were randomised to self-monitoring alone, self-monitoring with telemonitoring, or usual care, and general practitioners titrated medication against home readings. At 12 months, systolic blood pressure was 3.5 mmHg lower with self-monitoring and 4.7 mmHg lower with telemonitoring than with usual care (McManus et al., 2018). HOME BP extended this model by adding a patient-facing digital interface with lifestyle feedback and costed the intervention at roughly £11 per mmHg reduction (McManus et al., 2021). Both are strong evidence that measurement-plus-feedback can change a risk factor, but neither began from a risk equation, and neither used events as its endpoint.

A semi-closed loop adds behavioural interventions — text messages, app coaching, self-reported adherence — but lacks objective physiological feedback; the TEXT ME trial (Chow et al., 2015) and the Siopis et al. (2023) meta-analysis sit in this category. A full closed loop would require all four elements in one system: a validated risk equation that flags the individual, a triggered digital intervention, continuous home or wearable monitoring of the response, and a feedback mechanism that re-estimates risk and adjusts therapy, evaluated against hard cardiovascular events. No randomised trial of such a system has yet been published in primary prevention; this is the precise evidence gap.

Several considerations make this coupling natural rather than aspirational. First, the same data infrastructure — the smartphone, the EHR, the wearable — generates both the risk estimate and the intervention delivery, reducing friction. Second, dynamic risk models require repeated measurements, and digital interventions are precisely the mechanism that can collect those measurements while simultaneously acting on them. Third, the populations most poorly served by current risk equations — younger adults at long-term risk, patients with comorbidities, people in under-resourced health systems — are also the populations for whom scalable digital support is most promising.

The research agenda that follows from this view is concrete. Risk equations should be evaluated not only on discrimination and calibration but on whether the estimate they produce changes downstream intervention delivery. Digital interventions should be designed and tested in the populations that risk equations flag, with event-level endpoints and long follow-up. Calibration should be re-checked in every new population, rather than assumed from a derivation cohort. And the equity limitations of both tools — wearable bias, PRS underperformance in non-European groups, EHR missingness — should be addressed as first-order methodological problems rather than as implementation footnotes.

7. Discussion

The evidence reviewed here supports several bounded conclusions. The major contemporary risk equations — PCE, QRISK3, SCORE2, and PREVENT — all discriminate reasonably well, and each generation improves calibration by using more contemporary, more diverse, and richer data. The field has moved decisively away from race-specific coefficients and toward race-agnostic equations that incorporate renal function, social and clinical comorbidity, and longer time horizons. External miscalibration, however, remains the rule rather than the exception, and it is worst in precisely the populations — non-European ancestry, low- and middle-income settings, high-risk individuals — where prevention is most needed.

On the predictive-toolkit side, EHR-based machine learning consistently improves discrimination and extends risk estimation to patients excluded by fixed equations, but its transportability across health systems remains under-tested. Wearables, exemplified by the Apple Heart Study, can detect asymptomatic arrhythmia at scale, but their user base is self-selected and their absolute yield per wearer is low. Polygenic scores carry real genetic signal but have not yet demonstrated that they change individual treatment decisions in routine practice, and their performance in non-European populations is a live equity concern. The most defensible position is that none of these tools has yet replaced the classical risk equation; rather, each is a candidate addition whose incremental value depends on the decision context and the population.

On the intervention side, digital health — text messaging, smartphone apps, connected home devices, telehealth — produces modest but reproducible reductions in systolic blood pressure, LDL cholesterol, and medication non-adherence, effects that are clinically meaningful at population scale even when small at the individual level. The limitations are also real: short follow-up, selected populations, high attrition, and a reliance on surrogate rather than event endpoints.

The central argument of this review is that the next methodological advance lies at the intersection. A risk score that flags an individual but does not trigger a tailored, adaptive, monitored intervention is incomplete; a digital intervention delivered without a baseline risk estimate and without a mechanism to re-assess the individual's response is inefficient. Contemporary epidemiology has the methods — dynamic prediction, causal inference, precision medicine, implementation science — to design and evaluate coupled systems, but the two research communities have not yet fully converged.

7.1 Equity and implementation

The coupling argument raises an equity problem that should be named rather than assumed away. Digital closed-loop systems require a smartphone, reliable connectivity, digital literacy, and willingness to share physiological data with an algorithm or a clinical team. These prerequisites are unevenly distributed: older adults, people with low health literacy, those in low- and middle-income countries, and groups with limited connectivity are systematically less likely to be reached by app-based interventions. The same digital divide that skews wearable user bases also skews the populations in whom closed-loop systems will be evaluated, raising the risk that the systems prove effective in already-advantaged groups while failing to reach the populations at highest absolute CVD risk.

The predictive tools carry their own equity gradients. Polygenic scores perform less well in non-European ancestral groups because genome-wide association training data are disproportionately European; EHR-derived models inherit the missingness and labelling bias of the health systems they draw on; and optical wearable sensors are less reliable in people with darker skin tones. A closed-loop system built on these tools could, without deliberate design, widen rather than narrow existing disparities.

Implementation research must therefore treat equity as a first-order design constraint: testing closed-loop systems in the populations they intend to serve, recalibrating models to local risk-factor distributions, and building offline and low-bandwidth delivery modes rather than assuming universal app access.

Regulatory and data-governance questions follow. Consumer devices are regulated as general wellness products in some jurisdictions and as medical devices in others, and the algorithmic feedback that a closed loop relies on may sit outside clinical oversight. Data privacy, the transparency of automated recommendations, and the liability of algorithm-driven medication titration are unresolved. A closed-loop system that improves population health but concentrates benefit in digitally engaged patients, or that deploys opaque algorithms without clinical accountability, would not be an epidemiological advance regardless of its C-statistic.

Several open disagreements deserve emphasis rather than resolution. Whether polygenic scores earn their place in routine prevention is not yet settled; the answer likely depends on whether future implementation trials demonstrate changed prescribing and improved event outcomes, not merely improved C-statistics. Whether machine learning on EHRs outperforms recalibrated classical equations after proper external validation remains contested, and the gap may narrow as classical equations are refreshed. Whether consumer wearable screening is cost-effective at population scale is unknown. These are not gaps that this review can close; they are questions that the next generation of prospective, pragmatic, event-endpoint trials should address.

8. Conclusion

Cardiovascular prevention has moved from a small set of cohort-derived coefficients to a diverse, data-rich, and increasingly digital landscape. The Pooled Cohort Equations, QRISK3, SCORE2, and PREVENT represent a trajectory of improving calibration, expanding variable sets, and growing attention to equity and longer time horizons. EHR-based machine learning, wearable sensors, and polygenic scores are extending what is measurable, and mHealth and telehealth interventions are demonstrating that behavioural and adherence support can be delivered at scale. The task now is not to build yet another marginal risk equation but to couple risk assessment with the digital interventions that act on it, to validate the coupled system in the populations it serves, and to measure whether the loop actually reduces cardiovascular events. The epidemiological tools exist; the implementation work remains.

CRediT authorship contribution statement

A.M.: Writing – original draft, Methodology, Formal analysis, Conceptualization. W.R.: Writing – original draft, Methodology. L.H.: Writing – review & editing, Methodology, Conceptualization.

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References

1. Chow, C. K., Redfern, J., Hillis, G. S., Thakkar, J., Santo, K., Hackett, M. L., Jan, S., Bompoint, S., Billot, L., Grobbee, R., Barqawi, G., Arrieta, A., & Webster, R. (2015). Effect of lifestyle-focused text messaging on risk factor modification in patients with coronary heart disease: A randomized clinical trial. *JAMA*, *314*(12), 1255–1265. https://doi.org/10.1001/jama.2015.10945
2. Goff, D. C., Jr., Lloyd-Jones, D. M., Bennett, G., Coady, S., D'Agostino, R. B., Sr., Gibbons, R., Greenland, P., Lackland, D. T., Levy, D., O'Donnell, C. J., Robinson, J. G., Schwartz, J. S., Shero, S. T., Smith, S. C., Jr., Sorlie, P., Stone, N. J., & Wilson, P. W. F. (2014). 2013 ACC/AHA guideline on the assessment of cardiovascular risk. *Circulation*, *129*(25 Suppl 2), S49–S73. https://doi.org/10.1161/01.cir.0000437741.48063.64
3. Hippisley-Cox, J., Coupland, C., & Brindle, P. (2017). Development and validation of QRISK3 risk prediction algorithms to estimate future risk of cardiovascular disease: Prospective cohort study. *BMJ*, *357*, j2099. https://doi.org/10.1136/bmj.j2099
4. Khan, S. S., Coresh, J., Pencina, M. J., et al. (2023). Novel prediction equations for absolute risk assessment of total cardiovascular disease incorporating cardiovascular-kidney-metabolic health: A scientific statement from the American Heart Association. *Circulation*, *148*(24), 1982–2004. https://doi.org/10.1161/CIR.0000000000001191
5. McManus, R. J., Mant, J., Franssen, M., Nickless, A., Schwartz, C., Hodgkinson, J., Davies, M., Ryan, V., English, P., Wang, J., Francis, D., Roderick, P. J., & Sutcliffe, P. (2018). Efficacy of self-monitored blood pressure, with or without telemonitoring, for titration of antihypertensive medication (TASMINH4): An unmasked randomised controlled trial. *The Lancet*, *391*(10124), 949–959. https://doi.org/10.1016/S0140-6736(18)30306-1
6. McManus, R. J., Little, P., Stuart, B., Morton, K., Raftery, J., Kelly, J., Bradbury, K., Zhang, J., Zhu, S., Murray, E., Jones, N., Ige, J., & Mair, J. (2021). Home and Online Management and Evaluation of Blood Pressure (HOME BP) using a digital intervention in poorly controlled hypertension: Randomised controlled trial. *BMJ*, *372*, m4858. https://doi.org/10.1136/bmj.m4858
7. O'Sullivan, J. W., Raghavan, S., Marquez-Luna, C., Luzum, J. A., Damrauer, S. M., Ashley, E. A., O'Donnell, C. J., Willer, C. J., & Natarajan, P. (2022). Polygenic risk scores for cardiovascular disease: A scientific statement from the American Heart Association. *Circulation*, *146*(5), e93–e118. https://doi.org/10.1161/CIR.0000000000001077
8. Patel, A. P., et al. (2023). A multi-ancestry polygenic risk score improves risk prediction for coronary artery disease. *Nature Medicine*, *29*(7), 1793–1803. https://doi.org/10.1038/s41591-023-02429-x
9. Perez, M. V., Mahaffey, K. W., Hedlin, H., Rumsfeld, J. S., Garcia, A., Ferris, T., Balasubramanian, V., Russo, A. M., Rajmane, A., Cheung, L., Hung, G., Lee, J., Waalen, J., Silva, C. T., Odisho, F., Maguire, M., & Talati, N. (2019). Large-scale assessment of a smartwatch to identify atrial fibrillation. *New England Journal of Medicine*, *381*(20), 1909–1917. https://doi.org/10.1056/NEJMoa1901183
10. Ridker, P. M., & Cook, N. R. (2016). The Pooled Cohort Equations 3 years on: Building a stronger foundation. *Circulation*, *134*(23), 1789–1791. https://doi.org/10.1161/CIRCULATIONAHA.116.025693
11. Siopis, G., Moschonis, G., Eweka, E., Jung, J., Kwasnicka, D., Yeboah-Asiamah Asare, B., Kodithuwakku, V., Willems, R., Verhaeghe, N., Annemans, L., Vedanthan, R., Oldenburg, B., & Manios, Y. (2023). Effectiveness, reach, uptake, and feasibility of digital health interventions for adults with hypertension: A systematic review and meta-analysis of randomised controlled trials. *The Lancet Digital Health*, *5*(3), e144–e159. https://doi.org/10.1016/S2589-7500(23)00002-X
12. Visseren, F. L. J., Mach, F., Smulders, Y. M., Carballo, D., Koskinas, K. C., Bäck, M., Benetos, A., Biffi, A., Boavida, J.-M., Capodanno, D., Cosyns, B., Czernin, J., Desormais, I., Di Angelantonio, E., Freiberger, E., Franco, O. H., Gonzalo Ordóñez, A., Giampaoli, S., Graeff, E., … Wegler, A. (2021). 2021 ESC Guidelines on cardiovascular disease prevention in clinical practice. *European Heart Journal*, *42*(34), 3227–3337. https://doi.org/10.1093/eurheartj/ehab484
13. Ward, A., Sarraju, A., Chung, S., Li, J., Harrington, R., Heidenreich, P., Palaniappan, L., Scheinker, D., & Rodriguez, F. (2022). Machine learning and atherosclerotic cardiovascular disease risk prediction in a multi-ethnic population. *npj Digital Medicine*, *5*, 150. https://doi.org/10.1038/s41746-022-00695-7